

**U.S. Department of Labor**

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**Issue Date: 21 July 2005**

Case No.: 2003-LHC-01184

OWCP No.: 5-70014

In the Matter of:

AVELINA SHEPPARD, widow of  
BARTEMUS SHEPPARD, JR.,  
Claimant,

v.

NEWPORT NEWS SHIPBUILDING AND DRY DOCK CO.,  
Employer.

Appearances:

Jennifer West Vincent, Esq.  
For Claimant

Jonathan Walker, Esq.  
For Employer

Before: DANIEL A. SARNO, JR.  
Administrative Law Judge

**DECISION AND ORDER**

This proceeding arises under the Longshore and Harbor Workers' Compensation Act ("LHWCA" or "the Act"), as amended, 33 U.S.C. §§ 901, *et seq.* Claimant, Avelina Sheppard, widow of Bartemus Sheppard ("Decedent"), is seeking widow's benefits pursuant to Section 9(a) and (b) of the Act. The parties stipulated that Claimant was exposed to asbestos dust during his employment from 1949 to 1959 at Newport News Shipbuilding and Dry Dock Company ("Employer").

A formal hearing was held on January 26, 2005, in Newport News, Virginia. The parties submitted stipulations, which were received into evidence and labeled as Joint Exhibit 1. At the

hearing Claimant submitted Exhibits 1 through 22, and Employer submitted Exhibits 1 through 15.<sup>1</sup> The parties submitted post-hearing briefs, and Claimant filed an additional response to Claimant's post-hearing brief. The findings and conclusions which follow are based on a complete review of the entire record in light of the arguments of the parties, applicable statutory provisions, regulations, and pertinent precedent.

### **ISSUE PRESENTED**

Did asbestos contribute to the development of Decedent's lethal lung cancer?

### **STIPULATIONS**

Employer and Claimant stipulated to, and I find, the following facts:

1. Claimant's decedent was employed by Newport News Shipbuilding and Dry Dock Company from 1949 until 1959 as a sheetmetal worker.
2. Throughout his employment at Newport News Shipbuilding and Dry Dock Company, Claimant performed work related to ship repair or ship construction aboard ships on the navigable waters of the James River or its adjacent piers and dry docks.
3. Newport News Shipbuilding and Dry Dock Company is in the business of construction and repairing oceangoing vessels and stipulates that this claim falls within the jurisdiction of the Longshore and Harbor Workers' Compensation Act.
4. Claimant's decedent was exposed to airborne asbestos dust fibers during and in the course of his employment with Newport News Shipbuilding and Dry Dock Company.
5. On March 23, 2000, Claimant's decedent was diagnosed with lung cancer.
6. On November 30, 2000, Claimant's decedent died of lung cancer.
7. Upon receipt of the knowledge of the diagnosis of death, as a result of asbestos-related lung cancer the Claimant's decedent gave timely notice of his injury to the Employer and filed a timely claim for benefits under the Longshore and Harbor Worker's Compensation Act; and the Employer filed a timely controversion.

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<sup>1</sup> The following abbreviations will be used as citations to the record:

CX – Claimant's Exhibit

EX – Employer's Exhibit

JX – Joint Exhibit

Tr. – Transcript of the hearing

8. The Claimant married the decedent August 21, 1981, and she remained married to, resided with, and was dependent upon decedent until his death.
9. The weekly wage applicable at the time of decedent's death is \$466.91, the National Average Weekly Wage.

## **SUMMARY OF THE EVIDENCE**

### **Dr. John Holter**

Dr. John Holter is an Assistant Professor of Medicine in the Pulmonary Division of East Carolina University in Greenville, North Carolina. He examined Decedent on May 2, 1986. (CX 7). In his report, Dr. Holter stated that Claimant was a 40 pack-a-year smoker who claimed to have had significant asbestos exposure during his employment at Newport News Shipbuilding from 1949 to 1959. (CX 7 at 1). Dr. Holter reviewed Decedent's old chest x-rays from February 22, 1984; May 21, 1983; and March 14, 1981. He also reviewed a film from March 27, 1984, which Dr. Holter found to be remarkable for diffuse interstitial nodular changes throughout the lungs. (*Id.*). On the PA film, Dr. Holter stated that there was a subtle suggestion of some pleural plaques and a definite area of pleural thickening in the minor fissure, laterally, as well as pleural thickening on the lateral film, posteriorly. (CX 7 at 2). Dr. Holter found no dominant area of parenchymal activity. (*Id.*).

Dr. Holter conducted a physical examination of Decedent, which was unremarkable except for decreased breath sounds over the lungs, scattered bibasilar rales, and an increased forced expiratory time. (CX 7 at 2). Additionally, Dr. Holter noted a questionable presence of early clubbing in the fingernails. (CX 7 at 2).

Dr. Holter then proceeded with a flexible fiberoptic bronchoscopy. He indicated that a bronchoscopy was necessary because Decedent had a high risk for bronchogenic carcinoma due to his asbestos exposure and smoking history. (CX 7 at 2). The bronchoscopy was remarkable for diffuse bronchotic changes. The right middle lobe was then lavaged with 160cc of saline and was "remarkable for the presence of ferruginous bodies characteristic of asbestosis." (CX 7 at 3). Dr. Holter concluded his report by stating:

My impression is that Mr. Sheppard's pulmonary symptoms are due to a combination of occupational lung disease and chronic bronchitis secondary to cigarette smoking. His occupational exposure is remarkable for both asbestos and silica. It is very possible that the questionable nodal activity in the mediastinum on gallium scan is due to granulomatous reaction secondary to silica exposure.

(CX 7 at 3).

**Dr. Allan Smith**

Dr. Allan Smith is a pathologist at Beaufort County Hospital in Washington, North Carolina. (EX 1). Dr. Smith examined and interpreted the tissue obtained from Decedent's fine needle aspiration on March 23, 2000. (*Id.*). Dr. Smith described his findings as follows:

This very cellular specimen is composed of numerous cohesive clusters of markedly atypical glandular cells with high N/C/ ratios, prominent nucleoli, a delicate cytoplasm and marked nuclear pleomorphism. No squamous features of neuro-endocrine features are identified.

(EX 1).

**Dr. Jacques Legier**

Dr. Jacques Legier is a pathologist at Riverside Regional Medical Center in Newport News, Virginia. Dr. Legier conducted a surgical pathology consultation report at the request of Claimant's counsel. (EX 3). He received one slide labeled FNA00-20 from Beaufort County Hospital in Washington, North Carolina. Dr. Legier's report stated:

This 73 year old man died on 11/30/00 after employment as a sheetmetal worker and metal smith at the shipyard where he had significant asbestos exposure. It was also noted that Decedent smoked from 1942 to 1987, less than one-half pack per day; and that a clinical diagnosis of asbestosis had been made in 1986.

The cytology smear shows abundant malignant cells, which include frequent giant or multinucleate forms indicating an anaplastic large cell carcinoma with giant cell features, usually a variant of poorly differentiated adenocarcinoma.

There is no clinical evidence of primary carcinoma in other sites thus, the lesion appears to arise in the lung.

A previous report (XC-86-0230) had found asbestos bodies in a lavage specimen. An x-ray report showed evidence of probable pleural plaques.

I conclude, with reasonable degree of medical certainty, that Mr. Sheppard had unquestioned malignancy of the lung (giant cell adenocarcinoma), which is his cause of death and has occurred as a complication of his occupational exposure to asbestos, which is supported by his occupational history, previous visualization of asbestos bodies in his bronchial lavage and radiological evidence of pleural plaques.

(EX 3).

**Dr. John Maddox**

Dr. John Maddox is a board certified pathologist who currently practices at Riverside Regional Medical Center in Newport News, Virginia. (CX 5). Dr. Maddox has co-authored a few articles on asbestos-related disease since joining Riverside Medical Center in 1982. He noted that publication of articles is entirely optional as Riverside is a community hospital, not an academic institution. (CX 6 at 5). Dr. Maddox often reviews litigation cases dealing with lung disease; he stated that approximately 90 percent of the cases he sees are from plaintiff's firms; the other 10 percent are from defense firms. (*Id.*).

In his report dated April 7, 2003, Dr. Maddox reviewed a smear labeled FNA-00-20 and derived from Decedent. Dr. Maddox found clumps and clusters of large malignant cells, but he did not detect any asbestos bodies. Dr. Maddox did not have Decedent's alveolar lavage for review. Dr. Maddox concluded:

According to the criteria of Roggli, Greenberg and Pratt . . . "[T]he weight of the evidence at this time seems to indicate that, in an asbestos worker with carcinoma of the lung who also smokes cigarettes, asbestosis must be present clinically or histologically (or there should at least be a tissue asbestos burden content within the range of values observed in patients with asbestosis) in order to assign a substantial contributing role to asbestos in the causation of the lung cancer."

With a reasonable degree of medical certainty:

- based on the cytology, this is a poorly differentiated adenocarcinoma.
- Based on the occupational exposure history, the description of ferruginous bodies and pleural plaques by Dr. Holter, the patient had an increased pulmonary asbestos burden.
- Based on the clinical diagnosis of asbestosis made by Dr. Holter, asbestos contributed to the development of this man's lethal pulmonary adenocarcinoma.

(CX 4 at 2).

Dr. Maddox also provided deposition testimony. (CX 6). In Dr. Maddox's opinion, the largest number of doctors subscribe to the theory that attribution of lung cancer to asbestos exposure can be linked by a diagnosis of histologic asbestosis or clinical asbestosis, or an asbestos burden that is high enough to support a diagnosis of asbestosis. On the other hand, Dr. Maddox believes that a small number of doctors subscribe to the more extreme theories; i.e. that any asbestos exposure is enough for attribution versus the requirement of a diagnosis of asbestosis before any attribution can be made. (CX 6 at 75-76). Dr. Maddox, citing a report of the Surgeon General, also noted that because Decedent had stopped smoking in 1987, his risk of developing lung cancer had dropped, and would have been similar to the same risk of the non-smoking population 10 years after he ceased to smoke. (CX 6 at 54).

In reviewing Decedent's pathology sample, Dr. Maddox explained that a fine needle aspiration ("FNA") enables a physician to withdraw material from a person's chest or lung so that a diagnosis of cancer or infection can be made. (CX 6 at 44). Dr. Maddox explained that if the tip of the needle can get at the edge of the tumor as to enable some of the fluids from the lung, it can be concluded that the patient has a high asbestos burden if those lung fluids contain a number of asbestos bodies. In comparison, a bronchial lavage is done to evaluate pneumoconiosis, and is generally handled through a filtration technique to completely recover all of the individual particles. (CX 6 at 47). Dr. Maddox noted that the technique of lung lavage is not as precise as the technique of lung tissue digestion and fiber counting. (CX 6 at 63).

Dr. Maddox was unable to determine from Decedent's FNA whether he had asbestosis. (CX 6 at 45). However, Dr. Maddox was able to make a conclusion of whether Decedent's asbestos exposure contributed to his lung cancer. (CX 6 at 46). Dr. Maddox concluded that Decedent's case satisfied the criteria of Roggli, Greenberg and Pratt, a textbook that he would rely upon in his pathology practice. (CX 6 at 48). Under the Roggli criteria, attributions of lung cancer to asbestos requires a diagnosis of asbestosis clinically or pathologically, or an asbestos burden high enough to support the diagnosis of asbestosis. (CX 6 at 48). Roggli's formula for asbestos bodies in iron stain tissue sections would be about 750 asbestos bodies per gram of wet lung tissue. (CX 6 at 57). It was not possible to calculate Decedent's tissue burden per Roggli's formula for the number of asbestos bodies in iron tissue stain sections because there were no such tissue samples available in this case. (CX 6 at 57).

Dr. Maddox stated that the Roggli criteria is essentially the same criteria as that used in the Helsinki document, which was published in the Scandinavian Journal of Work Environmental Health, a peer-reviewed journal. The Helsinki criteria was the result of a consensus conference held in 1997 and attended by 19 individuals - physicians of different types who were interested in asbestos-related disease. (CX 6 at 55). The conference participants constructed generally-accepted criteria for attributions of various disease processes to asbestos exposure. (*Id.*). The Helsinki criteria states that the "attribution of lung cancer to asbestos exposure must be supported by an occupational history of substantial asbestos exposure, or measures of an asbestos burden." (CX 6 at 50). In looking at asbestos burden, the Helsinki criteria require two or more asbestos bodies in tissue with a sectional area of one square centimeter, or a count of uncoated asbestos fibers that falls into the range recorded for asbestosis by the same laboratory. In terms of evaluating asbestos burden through a lung lavage, the Helsinki criteria require over one asbestos body per ml of lavage fluid.

In this case, although the lavage was done and counted by a light microscope, the concentration in numbers of asbestos bodies per milliliter was not stated in the Holter report. Dr. Maddox noted that Dr. Holter's 1986 report was written 11 years before the Helsinki criteria were published; therefore, it would have been impossible for Dr. Holter to anticipate these criteria. Although Dr. Holter did not quantify the number of asbestos bodies in the lavage fluid, he apparently found numerous asbestos bodies. In Dr. Maddox's opinion, this would satisfy the Helsinki criteria. (CX 6 at 57-58). Dr. Maddox remarked:

Well, in Dr. Holter's report, he states that the lavage fluid was remarkable for the presence of ferruginous bodies characteristic of asbestos. And then I'm skipping

some negatives, and then, my impression is that Mr. Sheppard's pulmonary symptoms are due to a combination of occupational lung disease. And I would understand that phrase to refer to the asbestosis that he has used just above that -- and chronic bronchitis secondary to cigarette smoking.

(CX 6 at 65).

Dr. Maddox acknowledged that when an asbestos body is seen in lavage fluid, it is not sufficient to make a diagnosis of asbestosis. (CX 6 at 67). However, Dr. Maddox relied on Dr. Holter's reliance on other criteria in his diagnosis of asbestosis, including physical exam findings, abnormal pulmonary function tests, chest x-rays that showed diffuse interstitial nodular changes throughout the lungs, and pleural plaques. (CX 6 at 68). It is important to Dr. Maddox that there be either a clinical diagnosis of asbestosis or an indication that there was an asbestos burden high enough to support a diagnosis of asbestosis. (CX 6 at 69). In this case, Dr. Maddox thought that both were present. Dr. Maddox emphasized that even if there were doubts about the sufficiency of Dr. Holter's conclusions that Decedent had asbestosis clinically, Dr. Maddox was satisfied that there was a high enough asbestos burden. (CX 6 at 69). Dr. Maddox noted that he generally does not base a high asbestos burden on exposure history alone. (EX 6 at 69). He explained that while he recognizes the validity of those criteria, he personally does not do that because it falls under the rubric of industrial hygiene. (CX 6 at 70).

#### **Dr. Paul Wheeler**

Dr. Paul Wheeler is an associate professor of radiology at the John Hopkins Medical Institutions in Baltimore, Maryland. (EX 9). Dr. Wheeler was asked by Employer to conduct a pneumoconiosis evaluation in this case. Dr. Wheeler received the medical records of Decedent, as well as copies of a PA and lateral chest x-ray taken on November 3, 2000 and a spiral chest CT scan taken on October 23, 2000. (EX 6 at 1). Dr. Wheeler stated that the chest film copies are suboptimal with scapulae overlying the periphery of upper lungs on the PA view. Additionally, several of the CT scans were stuck together and the emulsion damaged when the films were separated with about half the scans with lung settings partly damaged. Dr. Wheeler stated that he was happy to receive any additional good quality CT scans, but even without, the scans he received were enough to show that there was no pneumoconiosis or benign asbestos-related pleural plaques. (*Id.*). Additionally, Dr. Wheeler's report noted

The major abnormality is a mass in the left upper lobe involving pleura which was proven to be non-small cell lung cancer with giant cells on biopsy. The cancer metastasized to both adrenals, right more than left and there are masses in both lungs compatible with metastases or possible granulomata.

Other minor abnormalities include probable minimal healed TB in both apices with fibrosis, apical pleural thickening and a few nodules seen best on the CT

scans and there is minimal emphysema which is mainly centrilobular in the upper lobes also seen on the CT scan lung settings. Finally there is focal arteriosclerosis left coronary artery.

(EX 6 at 1).

#### **Dr. Mark Wick**

Dr. Mark Wick is a pathologist at the University of Virginia in Charlottesville, Virginia. (EX 8 at 1). He is board-certified in anatomic and clinical pathology. (EX 8 at 2). On March 15, 2003, Dr. Wick reviewed Decedent's medical records and a glass slide labeled TNA00-20. (EX 5). Upon review of the pathologic specimen, Dr. Wick encountered a large-cell undifferentiated carcinoma of the lung. Dr. Wick did not find any asbestos bodies in this specimen. (*Id.*). Dr. Wick concluded that there was no objective scientific proof for the premise that occupational asbestos exposure was responsible for the development of Decedent's lung cancer. (*Id.*). Dr. Wick noted that there were no radiographic findings that are diagnostic of asbestosis, a condition, which, in his opinion, is required before a causal attribution to asbestos can be made. (*Id.*). Dr. Wick stated that Decedent's 20+ year cigarette smoking history and emphysema put him at definite lifetime risk for the development of several malignancies, of which bronchogenic carcinoma was the principal representative. (*Id.*) Dr. Wick concluded to a reasonable degree of medical certainty that asbestos exposure had no role in the causation of Decedent's lung carcinoma. (*Id.*).

#### **Dr. Andrew Churg**

Dr. Andrew Churg is a board-certified pathologist at the University of British Columbia. (EX 15 at 3-5). Dr. Churg specializes in lung pathology, and has published nearly 300 papers, books, and chapters dealing with lung pathology. (*Id.*). Dr. Churg often reviews cases for parties involved in litigation. Half of the cases that come before Dr. Churg are for plaintiffs and half are for defendants. (EX 15 at 16).

Dr. Churg was asked to review the medical records and pathology materials of Decedent; he reached conclusions and wrote a report on May 5, 2001. (EX 15 at 5). Dr. Churg reviewed one pathology specimen labeled FNA-00-20. (EX 4). The specimen, which consisted of a fine needle aspirate of a lung mass, showed a nonsmall cell carcinoma of the lung. Dr. Churg explained that he believes that the association of asbestos exposure and lung cancer is the association of the specific disease, asbestosis, and lung cancer. He stated that the pathology specimen did not allow of evaluation of the presence or absence of asbestosis. (*Id.*). Dr. Churg explained that in this circumstance he must rely on radiologic findings, but as none were provided, he could not reach a conclusion about causation in this case. (*Id.*). Dr. Churg requested recent chest film and CT reports, or a B-reading.

Dr. Churg also provided deposition testimony. He explained that subsequent to writing his report, he received the report of Dr. Paul Wheeler, a radiologist who found no evidence of asbestosis in Decedent. On that basis, Dr. Churg concluded that there is no association between Decedent's exposure to asbestos fibers and his development of lung cancer. (EX 15 at 6). If

asbestosis was established radiologically, Dr. Churg would have concluded that an association existed. (*Id.*).

In Dr. Churg's opinion, there are three theories which deal with the association between asbestos exposure and lung cancer. One theory submits that any exposure, no matter how small, increases the risk of lung cancer; Dr. Churg states that there is no good data to support this theory, and in fact, there is a lot of data which discredits this theory. The second theory is premised upon the belief that exposure to asbestos at levels sufficient to produce asbestosis, even in the absence of asbestosis, are sufficient to produce an association between exposure and lung cancer. (EX 15 at 7). Dr. Churg explained that there is a lot of data which supports evidence of what constitutes a threshold level. However, Dr. Churg opined that the problem with this theory is that epidemiological studies do not show an increased lung cancer risk when there is not an increased asbestosis risk. (EX 15 at 7). Therefore, it is not just the exposure at the level of asbestosis that increases the lung cancer risk, but rather the actual presence of asbestosis that increases the risk. (*Id.*). Dr. Churg relied in part on a 1999 article by William Weiss in *Chest* magazine for his opinion. (EX 15 at 7-8). The third theory posits that asbestosis must be present in order for an increased lung cancer risk to exist. Dr. Churg believes that this theory is clearly supported by the epidemiological evidence. (EX 15 at 9). Dr. Churg noted that there is not a majority position in the medical community among these theories; he explained that the question of whether asbestos exposure is associated with increased lung cancer risk is one in which reasonable pathologists can differ. (EX 15 at 9-10).

Dr. Churg reviewed the 1986 report of Dr. John Holter. Dr. Holter's report noted that "films from 1984 show diffuse interstitial nodular changes throughout the lungs." Dr. Churg stated that while this finding could possibly represent asbestosis, it would be a peculiar finding because asbestosis is usually lower-zonal and not throughout the lung. (EX 15 at 11). Dr. Churg also explained that the ferruginous bodies that Dr. Holter described are not diagnostic of asbestosis; instead, these bodies only indicate that Decedent had been exposed to asbestos.<sup>2</sup> (*Id.*). Therefore, Dr. Churg is of the opinion that Dr. Holter could not have been sure that Decedent had asbestosis based on these test results. (EX 15 at 12). Dr. Churg noted that clinicians often mistake the word "asbestosis" with the phrase "asbestos exposure". (*Id.*). Dr. Churg explained that patients with asbestosis have a dominant area of parenchymal activity; Dr. Holter's report stated that there was "no dominant area of parenchymal activity." Dr. Churg noted that Dr. Wheeler did not find evidence of asbestosis on radiographic films from 2000. As asbestosis is a disease that does not go away, Dr. Churg explained that it is impossible for there to be interstitial markings that indicate asbestosis in 1984 but not in 2000. (EX 15 at 13).

Dr. Churg explained that it is possible to determine an asbestos fibre burden by looking at lung digests. Therefore, if he had received a lung digest from Decedent, Dr. Churg would have been able to analyze it and determine the asbestos fibre burden and if and how it fit into a scheme of disease. Dr. Churg explained that it was not possible for him to make such a determination in this case with the samples that were provided to him. (EX 15 at 19). In Dr. Churg's opinion, pathology diagnoses generally trump clinical diagnoses if the correct tissue has been sampled.

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<sup>2</sup> Additionally, Dr. Churg only *presumed* that the ferruginous bodies were asbestos bodies. (EX 15 at 11).

**Dr. Victor Roggli**

On September 16, 2004, Dr. Victor L Roggli, a pathologist at Duke University Medical Center in North Carolina, wrote in a response letter to Employer's counsel that "a pathologist should not, under currently established criteria, attribute fibrosis to asbestos exposure unless it is asbestosis, as defined by the CAP-NIOSH criteria. (EX 14).

**DISCUSSION OF FACTS AND LAW**

Section 9 of the Act provides for death benefits to certain survivors "if the injury causes death." 33 U.S.C. § 909. Where the immediate cause of death was not work-related, an eligible survivor may qualify for Section 9 benefits if the employee had a work-related medical condition that hastened his death. *See Fineman v. Newport News Shipbuilding & Dry Dock Co.*, 27 BRBS 104 (1993); *Woodside v. Bethlehem Steel Corp.*, 14 BRBS 601 (1982). Pursuant to Section 20(a) of the Act, it is presumed that a claimant's injury arose out of and in the course of his employment if the claimant can establish that he sustained a physical harm, and either that a work-related accident occurred or that work conditions existed which could have either caused the harm or aggravated a pre-existing condition. 33 U.S.C. § 920 (a); *Blake v. Bethlehem Steel Corp.*, 21 BRBS 49 (1988); *Jones v. J. F. Shea Co.*, 14 BRBS 207 (1981); *Keliata v. Triple Machine Shop*, 13 BRBS 326 (1981).

If the claimant meets this burden, the burden shifts to the employer to rebut the Section 20(a) presumption and to establish, with substantial countervailing medical or factual evidence, that the claimant's harm was not caused or aggravated by his employment. *Cairns v. Matson Terminals*, 21 BRBS 252 (1988); *Rajotte v. General Dynamics Corp.*, 18 BRBS 85 (1986). Once the presumption is overcome by the introduction of substantial evidence, the fact finder must evaluate all of the evidence and reach a decision based on the record as a whole. *Universal Marine Corp. v. Moore*, 31 BRBS 119 (4th Cir. 1997); *Devine v. Atlantic Container Lines, G.I.E.*, 25 BRBS 16 (1990); *Norat v. Universal Terminal & Stevedoring Corp.*, 3 BRBS 151 (1976). Claimant must show by a preponderance of the evidence that that Decedent's death was hastened by his occupational exposure to asbestos.

The parties have stipulated that decedent was exposed to airborne asbestos dust and fibers in the course of his employment at Newport News Shipbuilding. Claimant has also presented medical evidence showing that Decedent suffered from a lethal carcinoma of the lung. I find that Claimant has established a harm and the existence of working conditions which could have caused or aggravated that harm, and is entitled to the Section 20(a) presumption.

The burden now shifts to Employer to rebut the Section 20(a) presumption and establish with substantial evidence that Decedent's harm was not caused or aggravated by his employment. Employer offered the opinions of Drs. Churg, Wick, and Wheeler. Dr. Wheeler, a radiologist, did not find any evidence of asbestos-related pleural plaques on the films he

reviewed from Decedent, which were taken in 2000. Dr. Churg stated that Claimant did not have a clinical or pathological diagnosis of asbestosis nor did he have any asbestos burden which would be diagnostic of asbestosis. Dr. Wick noted that there were no radiographic findings that are diagnostic of asbestosis, a condition, which, in his opinion, is required before a causal attribution to asbestos can be made. Dr. Wick also did not find any asbestos bodies in the pathology specimen he reviewed, and emphasized that Decedent's smoking history put him at definite lifetime risk for the development of a lung carcinoma. Employer's evidence is sufficient to rebut the Section 20(a) presumption afforded to the Claimant.

As the presumption has been overcome by the introduction of substantial evidence, I must now evaluate all of the evidence and reach a decision based on the record as a whole. The parties seem to agree that a diagnosis of asbestosis is sufficient to attribute an individual's lung cancer to asbestos exposure. However, in the absence of asbestosis, the parties disagree as to whether attribution can be established in another manner. Claimant contends that asbestosis is not a *per se* requirement for attribution, which can also be established by a high asbestos burden. Employer disagrees with this contention, and instead argues that science has not concluded as a medical fact that a high asbestos burden is sufficient to attribute lung cancer to asbestos exposure. In support of their positions, the parties rely on the reports and deposition testimony of their respective expert physicians as well as an array of medical literature.

Given the agreement of the parties, the first question for the court is whether Decedent has asbestosis. The parties again agree that Decedent was never previously given a pathological diagnosis of asbestosis, nor was it possible for any of the expert pathologists in this case to make a pathological diagnosis of asbestosis. (EX 15 at 19; CX 7 at 59). The parties, however, dispute whether Decedent was ever given a clinical diagnosis of asbestosis. In Employer's opinion, Dr. Holter did not diagnose asbestosis in his 1986 report. Additionally, Employer notes that Dr. Wheeler, who examined Decedent's x-rays taken in 2000, did not diagnose asbestosis. Claimant disagrees, and argues that Decedent was diagnosed with asbestosis in Dr. Holter's 1986 report. Claimant cites Dr. Holter's notes indicating that Decedent had abnormal pulmonary function tests and x-rays which revealed diffuse interstitial fibrosis and pleural plaques. Additionally, Claimant notes that Dr. Holter observed asbestos bodies in Decedent's pulmonary lavage.

Dr. Holter's detailed report notes that he observed ferruginous bodies "characteristic of asbestosis". Dr. Holter ultimately attributed Decedent's symptoms to "a combination of occupational lung disease and chronic bronchitis secondary to cigarette smoking." Dr. Maddox understood the phrase "occupational lung exposure" to refer to the asbestosis that Dr. Holter used in his earlier statement. (CX 6 at 65). Claimant also argues that the diffuse interstitial nodular changes and the subtle pleural plaques observed by Dr. Holter are further support for inferring that he diagnosed asbestosis.

According to *Dorland's Medical Dictionary*, "ferruginous" means "containing iron or iron rust."<sup>3</sup> W. B. Saunders Co., *Dorland's Illustrated Medical Dictionary*, (28th ed. 1994).

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<sup>3</sup> The definition for "ferruginous bodies" is noted because Dr. Churg made a statement that he *presumed* that these bodies were asbestos bodies. (EX 15 at 11). Dr. Churg's statement suggests that the term "ferruginous bodies" does not necessarily denote "asbestos bodies". Nevertheless, as the expert physicians in this case seem to interpret Dr. Holter's observation of "ferruginous bodies" as "asbestos bodies", the court follows their interpretation.

Employer's expert Dr. Churg stated that the ferruginous bodies that Dr. Holter described are not diagnostic of asbestosis; instead, these bodies only indicate that Decedent had been exposed to asbestos. (EX 15 at 11). He then explained that many doctors mistakenly use the term "asbestosis" interchangeably with "asbestos exposure." Dr. Churg also stated that asbestosis is usually lower-zonal and not throughout the lung. (EX 15 at 11). Accordingly, Dr. Churg remarked that the diffuse interstitial nodular changes observed by Dr. Holter would be a peculiar finding if Decedent actually had asbestosis. Finally, Dr. Churg cited Dr. Holter's note of "no dominant area of parenchymal activity", which Dr. Churg explained usually exists when asbestosis is present. (EX 15 at 12-13).

Given the testimony of the experts and the statements in Dr. Holter's report, I do not agree with Claimant's position that Dr. Holter made a clinical diagnosis of asbestosis. Having already described his findings as "characteristic" of asbestosis, Dr. Holter could easily have diagnosed asbestosis if he was certain that it existed. However, he chose not to make this statement, instead attributing Decedent's symptoms to the all-encompassing term of "occupational lung disease". Because the term "occupational lung disease" is a generic term which includes a variety of various diseases, and due to the failure of Dr. Holter's other findings to sufficiently indicate the existence of asbestosis, I conclude that the evidence does not show that Dr. Holter made a clinical diagnosis of asbestosis.

Furthermore, there is no radiological diagnosis of asbestosis in this case. Dr. Wheeler, a radiologist at John Hopkins Medical Center, reviewed radiographic films taken of Decedent in 2000. Although Dr. Wheeler noted that the films he reviewed were not the best quality, he did not see any evidence of benign asbestos-related pleural plaques.<sup>4</sup> (EX 6 at 1). Because asbestosis is a progressive disease, as noted by Dr. Churg, it follows that Dr. Wheeler would have diagnosed asbestosis on Decedent's chest films from 2000 if Decedent actually had asbestosis already in 1984. Therefore, Dr. Wheeler's report further supports the finding that no diagnosis of asbestosis exists in this case.

In the absence of asbestosis, the remaining question is whether Decedent's lung cancer can be attributed to asbestos exposure alone. Claimant argues that asbestosis is not a *per se* requirement to associate a lung cancer with asbestos exposure. Dr. Maddox stated that if Dr. Holter's clinical diagnosis of asbestosis in this case was not sufficient, he felt that attribution still existed because of the asbestos bodies found by Dr. Holter in Decedent's lavage fluid. Employer argues that the association of lung cancer and asbestos exposure can only be made when the specific disease of asbestosis is present.

Before weighing the merits of either party's position on this issue, I will first look at whether the evidence of record meets the criteria offered by Claimant under this theory. Claimant argues that the evidence in this case satisfied the Roggli and Helsinki criteria.

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<sup>4</sup> Claimant urges the court to disregard the opinion of Dr. Wheeler, given Dr. Wheeler's comment that the x-rays were of poor quality and damaged. (Claimant Brief p.8). I disagree with Claimant's argument. Dr. Wheeler could have stated his findings without even mentioning the quality of the x-rays, or he could have explained the quality and chosen not to make findings at all. Instead, he was truthful about the quality of the x-rays he received and explained that he was still able to make some observations of the films. I find that Dr. Wheeler's candidness about the situation does not detract from the weight of his opinion, and accordingly, his report should be given the same weight as the other evidence in this case.

According to the Helsinki and Roggli criteria, in order to attribute a lung cancer to asbestos exposure, there should be either a clinical or pathological diagnosis of asbestosis, or an asbestos burden high enough to support the diagnosis of asbestosis. (CX 6 at 48). As already explained above, there is neither a clinical nor a pathological diagnosis of asbestosis in this case. Therefore, under the criteria offered by Claimant, Decedent must have an asbestos burden high enough to support a diagnosis of asbestosis in order to attribute his lung cancer to his asbestos exposure. According to Dr. Maddox, the Helsinki criteria require 2 or more asbestos bodies in a section area of 1 square centimeter of lung tissue. (CX 6 at 56-57). The Roggli criteria, likewise, require 100 asbestos bodies per gram of wet lung tissue. (CX 22 at 47). Dr. Maddox explained, however, that it was not possible to calculate Decedent's tissue burden per the Helsinki criteria or Roggli's formula because there were no tissue samples available in this case. (CX 6 at 57).

However, a lung lavage sample does exist in this case. To establish an asbestos fiber burden through a lung lavage, the Helsinki criteria require over one asbestos body per ml of lavage fluid. (CX 6 at 57). Dr. Maddox explained that Dr. Holter did not quantify the number of asbestos bodies he found, but, in textual terms, stated that "he found numerous or at least multiple asbestos bodies". (CX 6 at 57, 65). In Dr. Maddox's opinion, this would satisfy the Helsinki criteria. (CX 6 at 57-58). Dr. Maddox emphasized that because Dr. Holter's report was written 11 years before the Helsinki criteria were published, it was impossible for Dr. Holter to anticipate the standard which would result from the Helsinki conference. (CX 6 at 64). However, Dr. Maddox believes that based on Decedent's occupational exposure history, the description of ferruginous bodies in the lung lavage fluid and the presence of pleural plaques as seen by Dr. Holter, asbestos did contribute to the development of Decedent's lethal pulmonary carcinoma. (CX 6 at 58).

The court recognizes that Dr. Holter's report was written before the Roggli or Helsinki criteria were established for the counting of asbestos fibers. Nevertheless, Dr. Holter could have quantified the asbestos fibers he observed, even though the criteria for attribution did not yet exist. The evidence submitted by Claimant states that a quantified asbestos burden, as measured against the criteria set forth by the Helsinki conference and Dr. Roggli, is necessary to attribute a link between exposure and lung cancer. Specifically, the Helsinki criteria states: "analysis of lung tissue for asbestos fibers and asbestos bodies can provide data to supplement the occupational history." (CX 14 at 311-312). The criteria also state that "the attribution of lung cancer to asbestos exposure must be supported by an occupational history of substantial asbestos exposure or *measures* of fiber burdens."<sup>5</sup> (CX 14 at 314) (emphasis added). The Roggli text states that "because a few asbestos bodies can be found in the lungs of virtually everyone in industrialized nations, quantitative studies are required to draw inferences relative to exposure and various diseases processes." (CX 22 at 45).

As stated above, these texts established guidelines to identify persons with a high probability of exposure to asbestos dust at work, which includes the guidelines for lavage fluid. (CX 14 at 312). Even though Dr. Holter stated that "the lavage fluid was remarkable for the

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<sup>5</sup> With respect to the issue of using occupational history to attribute lung cancer to asbestos exposure, Dr. Maddox remarked that he would not base an asbestos burden on exposure history alone because "that's kind of an industrial hygiene type of argument that I don't usually comment upon." (CX 6 at 70).

presence of ferruginous bodies,” he did not frame his findings in a quantitative context; he neither identified the amount of bodies that he observed, nor did he state how much lavage fluid was contained in the sample he observed. The criteria of Roggli and Helsinki clearly require a quantified asbestos burden, not just the observation of asbestos bodies in tissue or samples. Although Dr. Maddox contends that Dr. Holter’s report meets the Helsinki and Roggli criteria, I find that the evidence of record – namely, Dr. Holter’s unmeasured observation of ferruginous bodies - is insufficient to establish that Decedent had a particular asbestos fiber burden which could serve as a basis to attribute the development of his lung cancer to his occupational asbestos exposure.

Finally, as Claimant failed to meet the criteria of the asbestos fiber burden theory under the Helsinki or Roggli criteria, it is not necessary to determine whether this is a reasonable and medically accepted theory for attributing lung cancer to asbestos exposure.<sup>6</sup>

### **CONCLUSION**

Decedent was neither clinically nor pathologically diagnosed with asbestosis, nor was an asbestos fiber burden established which would meet the standards set forth by the Helsinki or Roggli criteria to attribute lung cancer to asbestos exposure. Therefore, Claimant has not met her burden of proving by a preponderance of the evidence that Decedent’s lung cancer can be attributed to his asbestos exposure, and her claim for widow’s benefits must fail.

### **ORDER**

It is hereby **ORDERED** that Claimant’s request for widow’s benefits pursuant to Section 9(a) and (b) of the Act is **DENIED**.

SO ORDERED.

**A**

Daniel A. Sarno, Jr.  
Administrative Law Judge

DAS/jrr

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<sup>6</sup> The parties submitted numerous articles which support their respective positions on the issue of attribution. These articles were read and considered by the court and appear to be peer-reviewed and well-reasoned. Because it was not necessary, given the facts of this case, to determine which theory of attribution has merit, these articles are not discussed in detail in this Decision. However, the Helsinki and Roggli texts, identified as Claimant’s exhibits CX 14, CX 19, and CX 22, were discussed in detail because Claimant’s position relied heavily on these texts. This reliance is evidenced by Dr. Maddox’s thorough discussion of these texts in his deposition testimony. (CX 6).